Covid research & implications

Purpose: This document is a running curation of Covid research cited in scholarly journals and/or trustworthy popular media, with the goal of forming a story about the implications of this growing data set on personal and public health.

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Study	Published	Main takeaways	Notes
Post-acute COVID mortality			
Post-acute sequelae of COVID-19 in older persons: multi-organ complications and mortality Published: 13 june, 2023	Journal of Travel Medicine - Oxford Academic	Older adults (>60) with COVID-19 have a significantly higher risk of stroke, heart failure, coronary heart disease, myocardial infarction, interstitial lung disease, chronic pulmonary disease, neuropsychiatric outcomes, acute kidney disease, and all-cause mortality.	“Participants with vs without COVID-19 diagnoses had higher incidence and risk of all-cause mortality in both the UK and Hong Kong cohorts. Both were at significantly increased risk for cardiovascular, respiratory, neurologic, psychiatric, gastrointestinal, renal, and hepatic complications.”
Many excess deaths attributed to natural causes are actually uncounted COVID-19 deaths, new analysis reveals Published: Feb 6, 2024	ScienceDaily (via Boston University School of Public Health)	A new study provides the first concrete data showing that many of these excess deaths were indeed uncounted COVID-19 deaths. The study compared reported COVID-19 deaths to excess	"Our findings show that many COVID-19 deaths went uncounted during the pandemic. Surprisingly, these undercounts persisted well beyond the initial phase of the pandemic," says study

		found that increases in non-COVID excess deaths occurred at the same time or in the month prior to increases in reported COVID-19 deaths in most US counties.	BUSPH, who has led numerous studies analyzing excess mortality patterns and drivers during the pandemic.
Accelerated aging			
Persistence of spike protein at the skull-meninges-brain axis may contribute to the neurological sequelae of COVID-19 Published: Dec 11, 2024	(see description and reference under Brain Damage)		Approximately 400 million individuals may carry significant amounts of spike protein, which "may contribute to the long-term neurological effects of COVID-19 and Long COVID. This includes accelerated brain aging, potentially leading to a loss of five to ten years of healthy brain function in affected individuals."
Evidence for Biological Age Acceleration and Telomere Shortening in COVID-19 Survivors	International Journal of Molecular Sciences	Covid infection shortens the telomeres and ages the body by 5 years.	"The results show a consistent biological age increase in the post-COVID-19 population, determining a DeltaAge acceleration of 10.45 ± 7.29 years (+5.25 years above the range of normality)."
Shorter telomere lengths in patients with severe COVID-19 disease	Aging	Covid infection shortens the telomeres, which is a signal of shortened lifespan.	"Critically short telomeres impair the regenerative capacity of tissues and trigger loss of tissue homeostasis and disease."
Accelerated biological aging in COVID-19 patients	Nature Communications	DNA analysis shows people are biologically older than their age post Covid infection, regardless of previous health or severity of acute illness from Sars2 infection.	"We also find the increasing acceleration of epigenetic aging and telomere attrition in the sequential blood samples from healthy individuals and infected patients developing non-severe and severe COVID-19"
Immune system impairment			
Persistent SARS-CoV-2 infection: significance and implications	The Lancet *Infectious Diseases*	Persistent infection with SARS-CoV-2 might be more common than	"The formation of tissue reservoirs (including in non-respiratory tissues)

		persistent infection, but there is evidence that such infections can occur even in healthy individuals, sometimes with few or no symptoms.	new strategies for diagnosis and treatment.”
Immune damage in Long Covid Published: Jan 18, 2024	Science	SARS-CoV-2 infections can cause systemic immune cell activation and inflammation, widespread multiorgan dysfunction, and thrombosis.	“Not everyone fully recovers from COVID-19, leading to Long Covid, the treatment of which is a major unmet clinical need. Long Covid can affect people of all ages, follows severe as well as mild disease, and involves multiple organs.”
Persistent Risk of Developing Autoimmune Diseases Associated With COVID-19 Published: Jan 8, 2024	Journal of Clinical Rheumatology	COVID-19 is associated with an increased risk of developing autoimmune diseases, and this effect persists for a long time.	“The relative risk of any autoimmune disease was 2.32 (95% confidence interval, 2.08–2.60). All the investigated outcomes showed a significant risk associated with COVID-19, and the long-term risk is substantial for systemic sclerosis and immunoglobulin G4–related disease.”
SARS-CoV-2 and HIV-1: So Different yet so alike. Immune Response at the Cellular and Molecular Level	International Journal of Medical Sciences	SARS-2 and HIV-1 are similar in that they’re RNA viruses, believed to have zoonotic origins, and they both wreak havoc on the human immune system.	“COVID-19 is multiphasic in its pathogenesis, and includes a cytokine storm, lymphopenia and ARDS, which indicate poor prognosis. COVID-19 progression and severity are associated with a systemic dysregulation of the immune system and impaired type I IFN early responses.”
Reduction and Functional Exhaustion of T Cells in Patients With Coronavirus Disease 2019 (COVID-19)	Frontiers in Immunology	Covid infection is destroying and/or exhausting T cells which are a critical part of our immune system.	
AIDS and COVID-19 are two diseases separated by a	Curated at Researchgate	Covid is more like airborne AIDS than the flu in terms of its impact	“We observe a comparable reduction in B cells in both

			compared to AIDS patients”
Single-cell multiomics revealed the dynamics of antigen presentation, immune response and T cell activation in the COVID-19 positive and recovered individuals	Frontiers in Immunology	Covid infection is destroying naive T cells, suppressing B cell response, and triggering antigenic response (i.e. autoimmune response) which all serve to screw up an otherwise healthy immune system. This immune system damage is happening *after* acute infection, suggesting the body’s immune system is struggling to cope with Covid long after someone “recovers” from it.	“Importantly, we discovered that a subset of the naive T-cells from the healthy individuals ***were absent*** from the recovered individuals, suggesting a post-infection inflammatory stage.” (Emphasis added.)
SARS-CoV-2 Uses CD4 to Infect T Helper Lymphocytes	Curated at Medrixiv (pre-print)	Covid directly infects the cells, persists in bodily reservoirs, and uses the CD4 pathway to infect our lymphocytes, dysregulating our immune system.	“SARS-CoV-2 infected T helper cells express higher amounts of IL-10, which is associated with viral persistence and disease severity. Thus, CD4-mediated SARS-CoV-2 infection of T helper cells may explain the poor adaptive immune response of many COVID-19 patients.”
Superantigenic character of an insert unique to SARS-CoV-2 spike supported by skewed TCR repertoire in patients with hyperinflammation	Proceedings of the National Academy of Sciences (PNAS)	Covid is hyperactivating the body’s immune response in a similar way to bacterial superantigens, leading to things like MIS-C, cytokine storms, and toxic shock syndrome.	“Notably, the superantigen-like motif is not present in other SARS family coronaviruses, which may explain the unique potential for SARS-CoV-2 to cause both MIS-C and the cytokine storm observed in adult COVID-19.”
SARS-CoV-2 infection causes immunodeficiency in recovered patients by downregulating CD19 expression in B cells via enhancing B-cell metabolism	Nature	Covid infection causes lasting destruction to our immune system.	“We found that SARS-CoV-2 infection causes immunodeficiency in recovered patients”

initial mild-to-moderate SARS-CoV-2 infection		system from Covid infection. (Note that this study does not suggest things get better after 8 months - that's just the time horizon for the study.)	naive T and B cells and showed elevated expression of type I IFN (IFN-β) and type III IFN (IFN-λ1) that remained persistently high at 8 months after infection."
Transcriptional reprogramming from innate immune functions to a pro-thrombotic signature by monocytes in COVID-19	Nature	Covid-19 is messing with our monocytes, which are key white blood cells in our immune system, specifically reprogramming them to be "functionally pro-thrombotic" or prone to triggering blood clotting.	"We find that ex vivo isolated CD14+ monocytes from mild and moderate COVID-19 patients are phenotypically different from monocytes from healthy individuals."
Incident autoimmune diseases in association with a SARS-CoV-2 infection: A matched cohort study	British Medical Journal/ Yale	SARS-CoV-2 infection is associated with a 42.63% increased risk of developing new-onset autoimmune diseases after the acute phase of infection. (n = 641,704 patients with COVID-19)	This estimate was similar for common autoimmune diseases, such as Hashimoto thyroiditis, rheumatoid arthritis, or Sjögren syndrome. The highest IRR was observed for autoimmune disease of the vasculitis group. Patients with a more severe course of COVID-19 were at a greater risk for incident autoimmune diseases.
An Observational Study of Neurologic Function After COVID-19 Infection	National Institute of Neurological Disorders and Stroke (NINDS)	Deep phenotype analysis showed broad immune dysregulation was seen in the cerebrospinal fluid (CSF) of a small cohort of people with long COVID neurologic symptoms.	"The persistence of these immune abnormalities several months after a mild infection suggests the possibility of either a persistent infection or an aberrant immune response to the infection," Nath and co-authors observed.
Viral persistence, reactivation, and mechanisms of long COVID	eLife	This is a broad literature review attempting to understand the link between "long covid" and viral persistence in the body. It has a section about the connections to immune system impairment.	"Severe COVID-19 can result in decreased frequencies and functional exhaustion of antiviral cytotoxic lymphocytes, including cytotoxic T lymphocytes (CTLs) and natural killer (NK) cells, as well as diminished frequencies of plasmacytoid dendritic cells."

and T-lymphocyte senescence and increased monocyte activation correlating with increasing fatigue severity		study, regardless of severity of acute symptoms or lingering symptoms. “Immune abnormalities that were found in the entire group of long COVID patients were low grade inflammation (increased inflammatory gene expression in monocytes, increased serum pro-inflammatory cytokines) and signs of T-lymphocyte senescence (increased exhausted CD8+ TEMRA-lymphocytes).”	and persistent symptoms lasting up to one year after hospitalization. Increased fatigue severity associated with stronger signs of monocyte activation in long COVID patients and potentially point in the direction of monocyte-endothelial interaction. These abnormalities were present against a background of immune abnormalities common to the entire group of long COVID patients.”
Brain damage			
Persistence of spike protein at the skull-meninges-brain axis may contribute to the neurological sequelae of COVID-19 Published: Dec 11, 2024	Cell Host & Microbe	An AI-powered imaging technique that renders organs and tissue samples transparent uncovered previously undetectable distributions of spike protein in tissue samples from COVID-19 patients and mice. It found that the SARS-CoV-2 spike protein can persist in the brain’s protective layers (the meninges) and the skull’s bone marrow for up to four years after infection.	Approximately 400 million individuals may carry significant amounts of spike protein, which “may contribute to the long-term neurological effects of COVID-19 and Long COVID. This includes accelerated brain aging, potentially leading to a loss of five to ten years of healthy brain function in affected individuals.” “Vaccination reduced but did not eliminate spike protein accumulation after infection in mice.”
Long COVID is associated with severe cognitive slowing: a multicentre cross-sectional study Published: Jan 25, 2024	The Lancet *eClinicalMedicine*	Patients with Post-COVID-19 Conditions (PCC) showed pronounced cognitive slowing compared to age-matched healthy individuals who previously had symptomatic COVID-19 but did not manifest PCC.	“Cognitive slowing was evident even on a 30-s task measuring simple reaction time (SRT), with patients with PCC responding to stimuli ~3 standard deviations slower than healthy controls. 53.5% of patients with PCC's response speed was slower than 2 standard deviations from the control mean, indicating a high prevalence of cognitive slowing in PCC. This finding was

			fatigue, depression, anxiety, sleep disturbance, and post-traumatic stress disorder did not account for the extent of cognitive slowing in patients with PCC."
Microstructural brain abnormalities, fatigue, and cognitive dysfunction after mild COVID-19 Published: Jan 19, 2024	Nature Scientific Reports	SARS-CoV-2 affects the brain in individuals who did not require hospitalization, causing persistent fatigue, headaches, memory problems, and somnolence even 2 months after their COVID-19 diagnosis.	"Our results suggest persistent cognitive impairment and subtle white matter abnormalities in individuals mildly infected without anxiety or depression symptoms. The longitudinal analyses will clarify whether these alterations are temporary or permanent."
Para-infectious brain injury in COVID-19 persists at follow-up despite attenuated cytokine and autoantibody responses Published: Dec 22, 2023	Nature Communications	The blood of patients with COVID-19 show elevated markers of brain injury, both early on and months after a SARS-CoV-2 infection, particularly in those suffering from post-infection brain dysfunction or neurological diagnoses.	"...we show evidence of quantifiable neuroglial injury markers in blood from COVID-19 infection (...) These brain injury markers are associated with dysregulated systemic innate and adaptive immune responses in the acute phase of the disease, and suggest that these may represent targets for therapy."
MRI with generalized diffusion encoding reveals damaged white matter in patients previously hospitalized for COVID-19 and with persisting symptoms at follow-up Published: Oct 22, 2023	Brain Communications (Oxford Academic)	Advanced diffusion MRI showed differences in brain white matter structure between COVID-19 patients with long-term symptoms and healthy individuals.	The study involved 16 men previously hospitalized for COVID-19. "The observed changes, which are consistent with axonal damage, demyelination and oedema, might be a contributing factor to the diversity of central nervous system symptoms that many patients experience after COVID-19."
SARS-CoV-2 infection and viral fusogens cause neuronal and glial fusion that	Science Advances	COVID-19 can cause brain cells to fuse. Like wires connecting switches to the lights in	"Neuronal fusion is a progressive event, leads to the formation of multicellular syncytia,

		both the kitchen and bathroom lights at the same time, or neither of them. It's bad news for the two independent circuits. "In the current understanding of what happens when a virus enters the brain, there are two outcomes—either cell death or inflammation, but we've shown a third possible outcome, which is neuronal fusion."	fusion severely compromises neuronal activity." (…) "An important consideration is that fused neurons remain viable albeit with altered circuitry and function. This uncharacterized, difficult-to-detect event could explain some of the neurological consequences of viral infections of the nervous system."
Neurocognitive and psychiatric symptoms following infection with COVID-19: Evidence from laboratory and population studies	Brain Behavior & Immunity – *Health*	Not only did participants who previously had COVID-19 perform worse on two specific cognitive tasks, but brain imaging showed that during these tasks, there was a lack of oxygen reaching the sections of the brain that would normally be fully engaged.	"Converging findings from laboratory and population survey data support the conclusion that symptomatic COVID-19 infection is associated with task-related, functional imaging and self-reported indices of cognitive dysfunction as well as psychiatric symptoms."
Single-Cell Transcriptomics Reveals Evidence of Endothelial Dysfunction in the Brains of COVID-19 Patients with Implications for Glioblastoma Progression	Brain Sciences	COVID-19 infection can lead to an increased risk of Glioblastoma due to virus-induced endothelial dysfunction, an impairment of the cells that line the inner surface of blood vessels. Glioblastoma is a type of cancer that starts as a growth of cells in the brain or spinal cord. It grows quickly and can invade and destroy healthy tissue. Glioblastoma forms from cells called astrocytes that support nerve cells. There's no cure for glioblastoma.	"Brain cell type- specific altered expression of major genes involved in endothelial dysfunction in COVID-19 was observed, with KDR, NOS, TGFB1, ICAM1, EDN1, PROCR, ANGPT2, and PDGFB upregulated in endothelial cells of COVID-19 patients compared to the healthy control."
Is the Frontal Lobe the Primary Target of SARS-CoV-2?	Journal of Alzheimer's Disease	There's growing evidence that Covid infection attacks the brain's frontal lobe, which is responsible for	"...[T]here is some evidence that SARS-CoV-2 could preferentially and directly target the

		symptoms are reported in connection with Covid.	symptoms, fronto-temporal hypoperfusion on MRI, EEG slowing in frontal regions, and frontal hypometabolism on 18F-FDG-PET imaging."
SARS-CoV-2 drives NLRP3 inflammasome activation in human microglia through spike protein	Molecular Psychiatry (scholarly journal) IfIscience (reputable science news site)	COVID infection is causing the same inflammation that leads to Parkinson's, Alzheimer's and other forms of dementia.	Builds on a growing body of research that Covid directly interacts with microglia and triggers inflammatory response in the brain.
SARS-CoV-2 is associated with changes in brain structure in UK Biobank	Nature (scholarly journal) NY Times	Infection strongly correlated with (1) reduction in grey matter in the brain (2) brain tissue damage (3) reduction in overall brain size	UK-based study by a dozen+ researchers used MRI scans and controls for pre-existing conditions.
SARS-CoV-2 promotes microglial synapse elimination in human brain organoids	Molecular Psychiatry	Covid infection is likely destroying physical synapses in the brain.	This is an extremely worrisome impact of post-acute Covid.
COVID-19–associated Acute Hemorrhagic Necrotizing Encephalopathy: CT and MRI Features	Radiology & National Library of Medicine	This study from the very early days of the pandemic used brain scans to show that SARS-2 infection was associated with destruction of the brain's protective blood barrier and destruction of brain matter.	"Acute necrotizing encephalopathy (ANE) is a rare complication of influenza and other viral infections and has been related to intracranial cytokine storms, which result in blood-brain-barrier breakdown, but without direct viral invasion or parainfectious demyelination (3). Accumulating evidence suggests that a subgroup of patients with severe COVID-19 might have a cytokine storm syndrome (4)."
SARS-CoV-2 Infectivity and Neurological Targets in the Brain	Cell Mol Neurobiology	Because the SARS-CoV-2 virus infects human cells via the ACE-2 receptor, a very common reception point, the virus can do serious and persistent damage to any part of the body where ACE-2 cell receptors are present, including the brain.	"A real danger of SARS-CoV-2 infection is not only its highly transmissible and contagious nature and lethality but also: (i) SARS-CoV-2′s simultaneous and multipronged attack on many human cell- and tissue-types involving vital and critical

			systems; and (ii) an unprecedented coordinated disruption of the complex neurophysiology, neurochemistry and neurology of the cells of the brain that normally regulate these multiple neurobiological systems”
Assessment and Characterization of COVID-19 related cognitive decline: Results From A Natural Experiment Published: Nov 7, 2023 (Preprint)	MedRxiv (Preprint)	Covid-19 infection is linked to “significant decline in cognitive throughput”	“Cognitive decline worsened with coronavirus disease 2019 severity and was concentrated in participants reporting post-acute sequelae of SARS-CoV-2. (...) COVID-19 was most likely associated with the observed cognitive decline, which was worse among patients with PASC or severe COVID-19.”
Stroke			
Risk of ischemic stroke in patients recovered from COVID-19 infection: A systematic review and meta-analysis	European Stroke Journal	Recovered COVID-19 patients have a higher risk of ischemic stroke compared to subjects from the general population within 9 months from the index infection.	Study of 23,559,428 patients, of whom 1,595,984 had COVID-19; After 9.2 months, ischemic stroke had occurred in 4.40 out of 1000 patients survived to COVID-19 compared to 3.25 out of 1000 controls.
COVID-19 related stroke in young individuals	The Lancet Neurology	Covid causes clotting and increases the risk of stroke exponentially for people under age 50.	“[D]ata supporting an association between COVID-19 and stroke in young populations without typical vascular risk factors, at times with only mild respiratory symptoms, are increasing.”
Acute Ischemic Stroke During the Convalescent Phase of Asymptomatic COVID-2019 Infection in Men	JAMA	Covid infection doubles the risk of stroke in men under 50, with the average onset of stroke two months post-infection.	“Acute ischemic stroke could be part of the next wave of complications of COVID-19, and stroke units should be on alert and use serological testing, especially in younger patients or in

raises risk of heart attack, stroke	Health Publishing Research originally published in The Lancet	attack increases exponentially in the weeks and months after Covid infection.	appear to be especially risky, perhaps because they trigger an exaggerated inflammatory response that makes blood clots more likely.”
Risks of deep vein thrombosis, pulmonary embolism, and bleeding after covid-19: nationwide self-controlled cases series and matched cohort study	BMJ	People with Covid have a significantly elevated risk of blood clots, stroke, and bleeding for a long time after acute illness is over.	“Compared with the control period, incidence rate ratios were significantly increased 70 days after covid-19 for deep vein thrombosis, 110 days for pulmonary embolism, and 60 days for bleeding.”
Heart attack / heart disease			
SARS-CoV-2 infection triggers pro-atherogenic inflammatory responses in human coronary vessels	Nature Cardiovascular Research	SARS-CoV-2 infects coronary arteries, increases plaque inflammation, increasing risk of heart attack and stroke long after the acute infection period is over.	“SARS-CoV-2 induced a robust inflammatory response in cultured macrophages and human atherosclerotic vascular explants with secretion of cytokines known to trigger cardiovascular events. Our data establish that SARS-CoV-2 infects coronary vessels, inducing plaque inflammation that could trigger acute cardiovascular complications and increase the long-term cardiovascular risk.”
Excess risk for acute myocardial infarction mortality during the COVID-19 pandemic	Journal of Medical Virology	Before the pandemic, death by heart attack was dwindling. It has skyrocketed amid Covid, with younger people dying of heart attacks. Strong correlation to Covid infection.	“The trend of mortality suggests that age and sex disparities have persisted even through the recent Omicron surge, with excess AMI-associated mortality being most pronounced in younger-aged adults.”
Long-Term Adverse Effects of Mild COVID-19 Disease on Arterial Stiffness, and Systemic and Central Hemodynamics: A Pre-Post Study	Journal of Clinical Medicine	Even mild cases of COVID can lead to changes in your arteries which can cause an increased chance of heart attacks and strokes;	Various parameters of arterial stiffness and central hemodynamics responded to the mild COVID-19 disease in predominantly healthy individuals. The worsening of measured

		prone to dysfunction”	process in the vasculature following the infection.
COVID and the Heart: It Spares No One	Johns Hopkins	Covid infection does tremendous damage to the endothelial layer of our blood vessels and reprograms some white blood cells to induce clotting instead of fighting pathogens. These problems persist well beyond the acute illness period and may cause lasting damage.	“[P]eople with COVID-19 have a higher risk of all sorts of heart problems at one year. That included arrhythmias (irregular heart beats or the heart beating too fast or too slow) and atrial fibrillation (a fast heart rhythm in a particular pattern). We found evidence of an increased risk of stroke, of blood clots in the legs and the lungs, and of heart failure and heart attacks.”
Objective Hemodynamic Cardiovascular Autonomic Abnormalities in Post-Acute Sequelae of COVID-19	Canadian Journal of Cardiology	Even non-hospitalized “mild” COVID-19 infections can result in long-term cardiovascular autonomic abnormalities (CAA)	Most common was initial orthostatic hypotension hemodynamic (IOH40). Women had increased frequency of postural orthostatic tachycardia syndrome hemodynamic (POTSHR), but IOH40 was equally prevalent between sexes.
Prior COVID-19 infection is associated with persistent and higher thrombus burden in acute coronary syndromes	European Heart Journal	Covid infection is wrecking the endothelium, a critical membrane layer that lines the heart and blood vessels, resulting in higher risk of a wide range of cardiovascular problems.	“Our findings suggest that COVID-19 infection may lead to persistent endothelial dysfunction and hypercoagulability, portending increased severity of coronary artery ectasia and coronary thrombosis even after recovery from the initial infection.”
Chronic Fatigue			
Differential Cardiopulmonary Hemodynamic Phenotypes in PASC Related Exercise Intolerance	European Respiratory Journals	COVID disrupts the body’s capacity to have energy on a cellular level: “...despite the fact that the heart was pumping oxygenated blood that the lungs were providing adequate	This study highlights the importance of considering impaired pEO_2 in PASC patients with persistent exertional intolerance unexplained by conventional investigative testing. Results of current study

			PASC with a primary peripheral limitation to exercise.
Estimated Global Proportions of Individuals With Persistent Fatigue, Cognitive, and Respiratory Symptom Clusters Following Symptomatic COVID-19	JAMA (Journal of the American Medical Association)	Even mild COVID-19 cases can have major and long-lasting effects on people's health; This multi-country study found that 90% of people living with long COVID initially experienced only mild illness with COVID-19.	Long-term symptoms included fatigue, shortness of breath and cognitive problems such as brain fog – or a combination of these – that affected daily functioning. These symptoms had an impact on health as severe as the long-term effects of traumatic brain injury.
Persistent Exertional Intolerance After COVID-19: Insights From Invasive Cardiopulmonary Exercise Testing	Chest Journal (American College of Chest Physicians)	Patients who recovered from mild COVID-19 continue to exhibit persistent exertional limitation that often is unexplained by conventional investigative studies.	Even patients without cardiopulmonary disease demonstrate a marked reduction in peak VO2 from a peripheral rather than a central cardiac limit, along with an exaggerated hyperventilatory response during exercise.
The Relationship between Physical Activity and Post-Acute COVID-19 Syndrome	International Journal of Environmental Research and Public Health	Almost half of those surveyed were no longer able to complete activities of daily living (ADL) without assistance. 74.84% of participants reported the effect of physical activity (PA) worsened their symptoms.	"Physical activity's potential benefits should be balanced against adverse effects, including the worsening of Long COVID (LC) symptoms. Policymakers need to consider the multitude of effects LC can have at both the individual and societal level."
Chronic Fatigue and Post-exertional Malaise Following a COVID Infection: An Observational Study	Physical Therapy & Rehabilitation Journal	COVID-19-induced post-exertional malaise (PEM) is common, and makes the road to recovery challenging because of the potential for setbacks and deteriorated function following any overexertion.	"Post-exertional symptom exacerbation must be considered by physical therapists working with people recovering from COVID-19, and rehabilitation needs to be carefully designed based on individual presentation."
Altered mitochondrial respiration in peripheral blood mononuclear cells of post-acute sequelae of SARS-	ScienceDirect (via Mitochonrian)	Some who suffer from "long-covid," a vague term for a wide range of post-acute symptoms lingering after SARS-2	"Mitochondrial dysfunction has been extensively studied in acute SARS-CoV-2 infection. (Romão et al.,

		ability to produce ATP (energy).	dysfunction is partly due to the extensive viral targeting of mitochondria and mitochondrial proteins."
Impact on kids			
Postacute Sequelae of SARS-CoV-2 in Children Published: Feb 7, 2024	Pediatrics *American Academy of Pediatrics*	Millions of people have long Covid, including children and pregnant people. While the severity and prevalence of COVID-19 in the pediatric population were not as high as in adults, PASC does entail severe and long-lasting consequences, including the development of new autoimmune conditions and diabetes.	"Although most individuals recover within a few days or weeks from an acute infection, some experience longer lasting effects. (...) Children may develop conditions de novo, including postural orthostatic tachycardia syndrome, myalgic encephalomyelitis/ chronic fatigue syndrome, autoimmune conditions and multisystem inflammatory syndrome in children."
Long COVID in Children and Youth After Infection or Reinfection with the Omicron Variant: A Prospective Observational Study	The Journal of Pediatrics	Reinfection from Omicron can lead to Long COVID in children and youth - even when a first SARS CoV-2 infection did not.	"A substantial numbers of children are likely to be impacted".
Covid-19 is a leading cause of death in children and young people ages 0-19 years in the United States Published: May 10, 2023	Curated in medrixv	Contrary to popular narratives, Covid has been a leading cause of death for young people since the pandemic began, and it was especially bad between the springs of 2021 and 2022.	"Due to the impact of mitigations such as social distancing and our comparison of a single disease (Covid-19) to groups of causes such as deaths from pneumonia and influenza, these rankings are likely conservative lower bounds."
Post-COVID-19-associated morbidity in children, adolescents, and adults	PLOS Medicine	Covid significantly increases the chance of death in children, adolescents, and young adults across multiple risk areas.	"We observed significant new onset morbidity in children, adolescents, and adults across 13 prespecified diagnosis/symptom complexes, following COVID-19 infection."
On the role of different age groups in propagating Omicron	BMJ	Kids *do* spread Covid, and in fact were the largest vectors of	"Children aged 10-19y played the greatest relative role in

			children aged 0-9y and adults aged 20-29y, as well as adults aged 30-49y."
Delta and Omicron killed far more children than flu ever does.	Inside Medicine	Covid is killing kids at 6x the rate of seasonal flu.	"Make no mistake: Covid-19 is imparting an unusual burden on children compared to all other respiratory viruses."
Multisystem inflammatory syndrome in children (MIS-C) showing disseminated aspergillosis, cytomegalovirus reactivation and persistent SARS-COV-2: Case report with autopsy review	Pathology Research and Practice	Some kids, probably about 1%, who are infected with Covid experience severe autoimmune responses like MISC-C and "cytokine storm," which can be fatal.	"Although MIS-C is generally considered to be a post-infectious hyperimmune reaction, persistence of SARS-COV-2 is a feature in all autopsies of MIS-C patients reported to date, suggesting a possible role in the pathogenesis, at least in fatal cases."
Chronic lung disease in children due to SARS-CoV-2 pneumonia: Case series	Pediatric Pulmonology	The study's authors look at about a half-dozen children with Covid pneumonia to try to understand the virus' impact on their respiratory systems.	"There is very little information on the long-term treatment of post-SARS-CoV-2 lung disease, especially in young children. Children with SARS-CoV-2 infection mostly have mild symptoms with little to no long-term sequelae. Mortality and severe disease have been reported in young infants especially in LMIC."
T Cell Cross-reactivity in Autoimmune-like Hepatitis Triggered by COVID-19	Science Direct	A major upswing in pediatric hepatitis was theorized to be cause by adenovirus, but this research strongly suggests Covid-19 infection in kids is the culprit and identifies the mechanism for induced hepatitis.	"[O]ur findings support the notion that cross-reactivity of clonally expanded T cells could be one of the causes of COVID-19-related autoimmune-like hepatitis, including pediatric hepatitis of unknown etiology."
Association of COVID-19 with respiratory syncytial virus (RSV) infections in children aged 0–5 years in the USA in 2022: a multicentre retrospective cohort	Family Medicine and Community Health	Covid infection increased the risk of secondary RSV infection in kids 0-5 by nearly 50% and was likely the primary driver of the 2022 "tripledemic" of Covid,	"COVID-19 was associated with a significantly increased risk for RSV infections among children aged 0–5 years in 2022. Similar findings were replicated for a study

			contributed to the 2022 surge of RSV cases in young children through the large buildup of COVID-19-infected children and the potential long-term adverse effects of COVID-19 on the immune and respiratory system.”
Diabetes			
Diabetes risk rises after COVID, massive study finds	Nature News (but the study was published in The Lancet Diabetes & Endocrinology)	Huge study, 180k people, showed that risk of new onset diabetes goes up significantly after even mild Covid infection, with more severe acute disease increasing the odds of diabetes considerably.	These same researchers used a similar methodology to show strong connection between Covid infection and kidney disease, heart failure, and stroke.
Association of COVID-19 with diabetes: a systematic review and meta-analysis	Nature	This meta analysis looked at eight studies and found that Covid infection has a strong correlation to new onset diabetes.	“In this systematic review and meta-analysis, COVID-19 was associated with higher risk for developing new onset diabetes among survivors. Active monitoring of glucose dysregulation after recovery from severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection is warranted.”
Sexual health			
Investigation of the effect of COVID-19 on sperm count, motility, and morphology	Journal of Medical Virology	Men with Covid have reduced sperm count, suggesting the virus and subsequent disease impact male reproductive health.	“It was observed that men with COVID-19 had decreased sperm concentrations suggesting that COVID-19 may have a negative effect on male fertility.”
Tip of the iceberg: erectile dysfunction and COVID-19	Nature	Covid is likely contributing to male erectile dysfunction by destroying the endothelial layer of key blood vessels (which is a known Covid impact throughout the body) and directly damaging	“In summary, COVID-19 infection could affect male sexual function through endothelial damage in erectile tissue, testicular damage, and psychological alterations.”

decline even after mild COVID infections Published: June 26, 2023	CIDRAP, via original research by the Scientific Reproduction Unit in Madrid.	with COVID-19 have decreased sperm counts for more than 3 months following even mild infections, and the sperm they do produce is less able to swim."	intriguing because men produce new sperm every 2 or 3 months, and the findings are based on semen analyses taken after 100 days, suggesting COVID does long-term damage to the male reproductive tract."
Pregnancy & fetus development			
Maternal SARS-CoV-2, Placental Changes and Brain Injury in 2 Neonates Published: May 1, 2023	Pediatrics	Scientists studied two babies born to Covid-positive mothers who, despite testing negative themselves, showed signs of viral presence and a wide range of serious health problems, from seizures to brain injury to death in one instance.	"The constellation of clinical findings, placental pathology, and immunohistochemical changes strongly suggests that second-trimester maternal SARS-CoV-2 infection with placentitis triggered an inflammatory response and oxidative stress injury to the fetoplacental unit that affected the fetal brain."
Respiratory distress in SARS-CoV-2 exposed uninfected neonates followed in the COVID Outcomes in Mother-Infant Pairs (COMP) Study Published: Jan 24, 2024	Nature *Communications*	Babies born to mothers who contracted a COVID-19 infection while pregnant have "unusually high rates" of respiratory distress at birth or shortly after.	Maternal COVID-19 severity has been previously associated with poor infant outcomes although detectable SARS CoV-2 IgG levels are often present in neonates at birth
Developmental impairment in children exposed during pregnancy to maternal SARS-COV2: A Brazilian cohort study. Published: Dec 5, 2023	International Journal of Infectious Diseases	Study followed about 60 children of unvaccinated women infected with SARS-CoV-2 during pregnancy and compared to a control group to track neurodevelopmental issues, which were far more prevalent in those children whose mothers were infected during pregnancy. "SARS-CoV-2 exposure was associated with neurodevelopmental impairment, and specific guidelines are	"We followed 127 children for one year, 69 in the COVID-19-exposed Group (EG), and 68 in the control group (CG). All mothers were unvaccinated at the time of cohort inclusion and maternal demographics were similar in the two groups. 20.3% of EG children and 5.9% of the CG received a diagnosis of neurodevelopmental delay within 12 months of life (p=0.013, RR= 3.44; 95% CI, 1.19-9.95). For the exposed

		children's health."	months, 7% at 6 months, and 32.1% at 12 months." (Emphasis added.)
Comparison of Pregnancy and Birth Outcomes Before vs During the COVID-19 Pandemic	JAMA	Covid-19 is associated with increases in pregnancy-related complications and maternal deaths during delivery hospitalization.	"While live-birth outcomes and mode of delivery remained stable, small but significant increases in pregnancy-related complications and maternal death during delivery hospitalization were observed."
SARS CoV-2 infection as a risk factor of preeclampsia and pre-term birth. An interplay between viral infection, pregnancy-specific immune shift and endothelial dysfunction may lead to negative pregnancy outcomes	Annals of Medicine	SARS-CoV-2 having affinity to ACE-2 and causing it's downregulation receptor may cause endothelial injury leading to compliment activation and formation of NETs, together with RAS dysregulation this may cause preeclampsia to develop in pregnant patients.	PTB may occur in patients as an effect of SARS-CoV-2 infection in first or second trimester as an effect of TLR4 pathway dysregulation with lower levels of IFNβ.
Sex-Specific Neurodevelopmental Outcomes Among Offspring of Mothers With SARS-CoV-2 Infection During Pregnancy	JAMA	These findings suggest that male offspring exposed to SARS-CoV-2 in utero may be at increased risk for neurodevelopmental disorders.	This cohort study of 18 355 infants delivered after February 2020 found that male but not female offspring born to mothers with a positive SARS-CoV-2 polymerase chain reaction test result during pregnancy were more likely to receive a neurodevelopmental diagnosis in the first 12 months after delivery, even after accounting for preterm delivery.
Maternal SARS-COV-2 infection and prematurity: the Southern Michigan COVID-19 collaborative	The Journal of Maternal-Fetal and Neonatal Development	COVID-19 is an independent risk factor for preterm birth. The increased preterm birth rate in COVID-19 was primarily driven by medically indicated delivery, with preeclampsia as the principal risk factor. Symptomatic status and disease severity were significant drivers	The rate of prematurity was 8.9% in controls, 9.4% in asymptomatic cases, 26.5% in symptomatic COVID-19 cases, and 58.8% among cases admitted to the ICU.

among pregnant women with SARS-CoV-2 infection: an individual participant data meta-analysis		wide range of very poor outcomes for both mother and child.	infection at any time during pregnancy increases the risk of maternal death, severe maternal morbidities and neonatal morbidity, but not stillbirth or intrauterine growth restriction”